Effect of Passive Smoking on Asthmatic Children Who Have and Who Have Not Had Atopic Dermatitis*

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We studied 240 children with asthma who were themselves nonsmokers and had been referred consecutively to our clinic. They were aged 6 to 17 years. The severity of asthma was assessed by symptom score, by spirometry, and, in those who could perform the test reliably, by histamine bronchial challenge test. Those who reported having had a chronic or chronically relapsing itchy rash in characteristic locations were recorded as having had atopic dermatitis. Multiple analysis of variance revealed that children whose mothers smoked had significantly more severe asthma ($p<0.001$) but that atopic dermatitis had no apparent effect on the severity of asthma, either in its main effect ($p=0.71$) or in its interaction with maternal smoking ($p=0.66$). Although our previous study indicates that smoking mothers' children are more likely to develop asthma if they have had atopic dermatitis than if they have not, the severity of asthma does not appear to be associated with a history of atopic dermatitis. In smoking mothers, children who had had atopic dermatitis were just as severe as those who had not had atopic dermatitis as in those who had. *(Chest 1992; 101:16-18)*

MANOVA = multiple analysis of variance; PC_m = provocation concentration of histamine required to decrease FEV, by 20 percent

Smoke pollution in the home appears to aggravate symptoms in children with asthma. In our first study population, which comprised the asthmatic patients who attended our allergy clinic, we found that asthmatic symptoms were more severe, and that pulmonary function test results were lower in those whose mothers were smokers than those whose mothers were nonsmokers.[1-3]

Not only does passive smoking appear to exacerbate symptoms in those who are already asthmatic, there is also strong evidence that it causes asthma in certain children who would not otherwise have had this disease. This was found in a second study population, one that included all of the children who attended our allergy clinic. We demonstrated that children with atopic dermatitis were much more likely to have asthma if the mother smoked than if she did not smoke.[4] In those with no history of atopic dermatitis, by contrast, asthma was just as frequent in those with a nonsmoking mother as in those with a mother who smoked.

This finding, that passive smoking is a risk factor for causing asthma only in the children who have a history of atopic dermatitis, raises the question as to whether it is also solely asthmatic children with atopic dermatitis whose asthma is aggravated by smoke pollution in the home. Knowing the correct answer to this question is necessary to answer the following one: should smoking parents of an asthmatic child be counselled just as forcefully about smoke avoidance if the child has never had atopic dermatitis as when the child has had atopic dermatitis?

In order to answer these questions, we reanalyzed the data from our first study.[2] A history concerning the presence or absence of atopic dermatitis was available on all but three of the children who had comprised the earlier study's population.

Materials and Methods

The population and some of the methods were described in detail in an earlier publication.[1] In brief, the series included every child who had a history of asthma or frequent wheezing, who was aged 7 to 17 years, who had been referred to one of us (A.B.M.) at the Children's Hospital Allergy Clinic in Vancouver, and who was seen between Nov 1, 1983 and May 31, 1986. The data recorded at the first visit during this period were used in the study. There were 247 subjects in all.

Questionnaire

A trained interviewer put standardized questions to the patient and to the accompanying adult who, in 97 percent of the cases, was a parent. The parents or the accompanying person was asked how long the child had had the asthma or wheezing,[5] whether the child had suffered from a cold or respiratory infection during the preceding two weeks, and whether the child had received a medication which might affect the result of the histamine bronchial challenge test.[6] They were also asked how severe they considered the child's asthma to be, how frequently the child had wheezed and had received medication during the previous 12 months, whether corticosteroids had been given, and whether there was wheezing on exertion. Each of these features was assigned a numeric score indicating severity, and the sum of these numbers was the asthma symptom score.[1,2,6] In addition, parents were asked whether the child had ever had a rash and, if so, whether it had been itchy, how

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long it had lasted, and what its distribution had been. Only children who had had a rash which was itchy, which was chronic or chronically relapsing, and which had occurred in characteristic areas were classified as having had atopic dermatitis.[7]

When the previous questions had been completed, the parent or accompanying adult was asked whether the mother smoked and whether the father smoked.[3] The child was asked privately whether he or she smoked. The four children who admitted to being smokers were excluded from the study.

Forced Expiratory Spirogram

Forced expiratory maneuvers were performed until there were three in which the forced vital capacity (FVC) agreed within 5 percent. Three children who were unable to perform consistently satisfactory spirograms were eliminated from the series. The forced expiratory volume in one second (FEV_1) and the forced expiratory flow during the middle half of the FVC (FEF25-75%) were expressed as a percentage of the predicted mean for age, sex, and height.[8]

Bronchial Responsiveness to Histamine

Parents were contacted by telephone 48 hours prior to the appointment and were instructed to discontinue medications which might influence a bronchial challenge test. After the spirogram had been recorded, a bronchial challenge test was given to all of the 103 children who could perform the test reliably; that is, it was given to all except those who had recently taken medications which might interfere with the test[9] or had had a respiratory infection within the previous two weeks or had a FEV_1 which was less than 60 percent of predicted.

Aerosol histamine was administered by mask using the method of Cockcroft et al.[10] Children whose FEV_1 did not decrease by 20 percent when the strongest concentration (8 mg/ml) was administered were deemed, for the purpose of calculating the PC_{20}, to respond to double that concentration, *ie*, 16 mg of histamine acid phosphate per milliliter.

Statistical Analysis

There were 240 subjects, 206 of whom had data concerning all of the dependent and independent variables (other than PC_{20}, which was recorded in a subset of 103). Of the 206 subjects, 46 had smoking mothers, and 160 did not; 62 had had a recent respiratory infection, and 144 had not; and 79 had had atopic dermatitis, and 127 had not. Previous analysis of these patients[4] by multiple regression had indicated that the most important predictors of the severity of asthma were maternal smoking, recent respiratory infection, age, and the age of onset of asthma. A MANOVA test was therefore carried out using the following as dependent variables: FEV_1%; FEF25-75%; and asthma symptom score. The design consisted of a $2 \times 2 \times 2$ factorial analysis incorporating all possible interactions. The factors were whether or not the child had had atopic dermatitis, whether or not the mother smoked, and whether or not the child had had a recent respiratory infection. Age and the age of onset were used as covariates.

In a previous analysis, smoking by the father was found to be a predictor of the severity of asthma in boys only and not in the population as a whole; this variable was therefore not included in the MANOVA.[3]

The PC_{20} measurement was not included as a dependent variable because none of the children with a recent respiratory infection was given a histamine bronchial challenge test, the reason for omitting this test being that infection is thought to aggravate bronchial responsiveness.[9] Therefore, for PC_{20}, a separate two-factor analysis was run, with maternal smoking and atopic dermatitis as factors; and since age and the age of onset were not found to be predictive, analysis of variance, rather than analysis of covariance, was performed.

Table 1—*Adjusted Means of Measures of Severity of Asthma**

Measure	Maternal Smoking		Recent Respiratory Infection		Atopic Dermatitis	
	No	Yes	No	Yes	No	Yes
Asthma symptom score	6.9	9.1	7.4	8.6	7.9	8.1
FEV_1, percent of predicted	84.0†	76.5†	82.2‡	78.3‡	79.4§	81.2§
FEF25-75%, percent of predicted	71.0	59.2	68.9	61.3	62.8	67.4
ln PC_{20}‖	0.7¶	−0.1¶	...	...	0.4**	0.2**

*Adjusted for covariates, age and age of onset, and for two of the factors, maternal smoking, recent infection, and atopic dermatitis.
†$p = 0.001$.
‡$p = 0.008$.
§$p = 0.71$.
‖These means were adjusted by factors, maternal smoking and atopic dermatitis; no covariates were used.
¶$p = 0.008$.
**$p = 0.40$.

Results

When considering the main effects of the factors in the MANOVA analysis, children whose mothers smoked were found to have significantly more severe asthma than those whose mothers did not smoke ($p = 0.001$; see Table 1). In contrast, atopic dermatitis and recent respiratory infection had no apparent effect on the severity of asthma, either as main effects, or as a result of their separate or combined interactions with maternal smoking; however, the covariates, age and age of onset, were found to be highly significant predictors of the severity of asthma.

The analysis of the PC_{20} levels produced similar findings. The levels were significantly different in the children of the smoking mothers as compared with those of nonsmoking mothers ($p = 0.008$), but a history of atopic dermatitis was not predictive of the PC_{20} levels ($p = 0.404$).

Discussion

We had observed in another study that children in our clinic had asthma more frequently if the mother smoked than if she did not smoke, but that this increased frequency of asthma was limited to those who had had atopic dermatitis.[4] This finding raised the following question: was the aggravation of asthma in asthmatic children of mothers who smoked, as reported by us in our clinic patients[1-3] and confirmed by others in community surveys,[10,11] also confined to those who had had atopic dermatitis? It was in order to answer this question that we reanalyzed the information collected during our earlier study,[2] the one which had been performed on a clinic population of asthmatic children.

Our analysis reconfirmed that asthmatic children who had smoking mothers had more severe asthma

than those who did not, whether judged by the severity of symptoms or by pulmonary function testing, and it indicated that this aggravation of their asthma was not confined to those who had had atopic dermatitis; atopic dermatitis was not a predictor of the severity of asthma, either on its own, or combined with maternal smoking.

Our data did not confirm that recent respiratory infection was a predictor of increased severity of asthma. Even though the number of subjects was substantial, neither the main effect of recent infection, nor its interaction with atopic dermatitis or maternal smoking achieved statistical significance. Nevertheless, there is a possibility that in some subgroups of children, recent infection might be important in determining the severity of asthma.

It is not likely that children were incorrectly classified as nonsmokers themselves when they did in fact smoke. Using a carbon monoxide monitor, we have measured the CO content in the expired air of consecutive patients who were aged between 9 and 17 years and who were seen subsequently.[12] Only the two who admitted to being smokers had a CO concentration above 8 ppm; the other 77 who claimed to be nonsmokers all had a CO concentration below this level (unpublished data). These findings suggest that the children who attend our clinic are truthful about their smoking habits, as has been found in children in London[13] and New York.[14]

Although passive smoking may cause asthma only in children who have had atopic dermatitis,[4] we conclude that smoke aggravates asthma in those who have not had atopic dermatitis as well as in those who have. It is therefore appropriate to urge parents of all asthmatic children, even those who have not had atopic dermatitis, to refrain from smoking when in the house or when in the car with the child.

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